The Four Medial Ankle Tunnels: A Critical Review of Perceptions of Tarsal Tunnel Syndrome and Neuropathy

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KEYWORDS
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Evidence-based medicine is the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. The practice of evidence-based medicine means integrating individual clinical expertise with the best available external clinical evidence from systematic research. By individual clinical expertise we mean the proficiency and judgment that individual clinicians acquire through clinical experience and clinical practice… By best available external clinical evidence we mean clinically relevant research, often from the basic sciences of medicine, but especially from patient centered clinical research into the accuracy and precision of diagnostic tests (including the clinical examination)… Evidence-based medicine is not restricted to randomized trials and meta-analyses. It involves tracking down the best external evidence with which to answer our clinical questions. To find out about the accuracy of a diagnostic test, we need to find proper cross-sectional studies of patients clinically suspected of harbouring the relevant disorder, not a randomized trial. For a question about prognosis, we need proper follow-up studies of patients assembled at a uniform, early point in the clinical course of their disease.[1]

Numbness, burning, or tingling in the toes or the sole of the foot; nocturnal awakening with the foot tingling; worsening of symptoms as the day goes on; and cramping in the foot all are included in the symptom complex termed *tarsal tunnel syndrome* by Keck[2] and by Lam,[3] independently in 1962, and by those caring for this problem today. Keck and Lam each related their symptom complex to the carpal tunnel syndrome of the hand. For the tarsal tunnel syndrome, symptoms were reportedly relieved by dividing the flexor retinaculum. The flexor retinaculum joins the medial malleolus to the calcaneus to form the roof of the tarsal tunnel. Forming part of the wall and floor of the tarsal tunnel, safely covered by their own flexor sheaths, and without exposed synovium are the tibialis posterior, flexor digitorum longus, and flexor hallucis longus tendons. Only the posterior tibial artery and veins occupy the tarsal tunnel with the posterior tibial nerve. In the patient who has diabetes, instead of a space-occupying lesion creating extrinsic pressure on the posterior tibial nerve, metabolic abnormalities predispose the nerve to

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chronic compression. How to identify which patients have compression of the tibial nerve in the presence of neuropathy remains a point of controversy, as does the effect of surgical decompression of the tibial nerve and its branches in the patient who has neuropathy. It is the purpose of this review to identify the basic science and clinical evidence that can lead to improved quality of care and outcomes for patients who are otherwise viewed as having a progressive and irreversible medical problem.

MECHANISM OF COMPRESSION OF THE DISTAL TIBIAL NERVE

Pressure on a peripheral nerve of greater than 20 mm Hg is sufficient to reduce blood flow within the veins, and pressure greater than 40 mm Hg is sufficient to reduce blood flow within the arteries of that nerve.[4] At pressures greater than 80 mm Hg, structural changes occur that can cause irreversible damage to the nerve.[5] Ischemia of a peripheral nerve results in the symptoms of numbness and tingling referred to as paresthesias, and if the decreased oxygen content persists long enough, an ischemic conduction block of electrical activity in that nerve occurs. The pressure can come from a space-occupying lesion, such as a ganglion[6] or congenital anomalies (anomalous muscle[7] or high division of the tibial nerve[8]), posttraumatic or iatrogenic injury, or metabolic problems within the peripheral nerve itself that render it susceptible to chronic compression, such as decreased axoplasmic flow in diabetic and chemotherapy-induced neuropathy.[9,10] Positioning of the ankle joint may be responsible for decreasing the tarsal tunnel volume from a mean of 21.5 $\pm$ 0.9 cm^4 to 18.0 $\pm$ 0.9 cm^4 ($P < .001$) in full eversion or to 20.3 $\pm$ 1.0 cm^4 ($P < .001$) in full inversion (pronation or supination),[11] thereby increasing pressure on the tibial nerve in the tarsal tunnel from a mean pressure of 2 $\pm$ 1 mm Hg in neutral position to a mean of 32 $\pm$ 5 mm Hg ($P < .005$) in full eversion, or to a mean of 17 $\pm$ 5 mm Hg ($P < .05$) in full inversion. [12] Theoretically, relief of the pressure sufficiently soon permits complete restoration of nerve function.

If even minimal pressure persists about a peripheral nerve in the rat model for 2 months, there is a loss of endoneurial microvessel integrity, resulting in endoneurial edema.[13] If this pressure persists for 6 months in the rat[13] or primate model,[14] perineurial fibrosis and demyelination occur. With compression persisting for 12 months, there is further loss of demyelination and loss of large myelinated fibers.[13,14] Decompression of the nerve at this point results in restoration of large myelinated fibers but with incomplete remyelination.[14]

The flexor retinaculum, when viewed during surgery, is, however, almost always loose, such that increased pressure within the tarsal tunnel itself does not seem to be the mechanism possible for the ischemic symptoms of the tibial nerve and tarsal tunnel syndrome. This observation seems to explain why most reports from 1970 to 1996 of decompression of just the tarsal tunnel achieved excellent results in only 0%,[15] 15%,[16] 20%,[17] 24%,[18] 26%,[19] and 54%[20] of the reported patients in these retrospective level IV therapeutic studies. During this time, many investigators also continued to immobilize the ankle after the operation (**Table 1**).

Careful anatomic analysis demonstrates that the tarsal tunnel is not the equivalent of the carpal tunnel; rather, it is more closely the equivalent of the forearm. Therefore, the flexor retinaculum is equivalent to the distal forearm fascia. Relief of carpal tunnel syndrome would not occur if just the distal forearm fascia were to be divided. Careful anatomic analysis demonstrates that the thenar muscle origin from the transverse carpal ligament is equivalent to the abductor hallucis muscle arising from a thick ligament that begins immediately at the end of the tarsal tunnel. The tarsal tunnel ends when the flexor retinaculum splits to ensheathe this intrinsic muscle. Just as the hook process of the hamate divides the median nerve in its carpal tunnel from the ulnar nerve in its canal of Guyon, so does a thick septum go from the tendon sheaths or calcaneus to this ligament, creating a medial plantar tunnel and a lateral plantar tunnel. Just as there is a tunnel for the palmar cutaneous branch of the median nerve, so too is there at least one calcaneal tunnel, whose roof is part of the origin of the thick ligamentous roof of the medial and lateral plantar tunnels (**Fig. 1**).[21]

There are then four medial ankle tunnels. Is it possible that the sites of compression that give rise to the symptoms of tarsal tunnel syndrome are attributable to increased pressure within the medial and lateral plantar and calcaneal tunnels instead of within the tarsal tunnel itself? A recent systematic review of level IV retrospective clinical studies demonstrates increasing clinical outcomes related to the number of tunnels decompressed (see **Table 1**).[22]

A recent study of the pressures within the medial and lateral ankle tunnels, and changes in these pressures related to ankle position, demonstrated that the pressures within the medial and lateral plantar tunnels increased significantly higher than in the tarsal tunnel.[23] For example, the medial plantar tunnel pressure increased from a mean of

Table 1
Summary of "Therapeutic Level IV" tarsal tunnel syndrome studies

Study Date	No. Patients	NCV EMG[a]	Tinel Positive	Tunnels Released	Immobilized After Surgery	Results (%)			
						Excellent	Good	Fair	Poor
1970, Linscheid et al	24	100%	Yes	1	NA	0	50	30	20
1974, Mann	9	90%	Yes	3	3 weeks	78	0	11	11
1989, Stern and Joyce	15	40%	Yes	4	10 days	54	20	20	6
1992, Byank and Curtis	49	100%	Yes	1	NA	26	53	53	9
1993, Sammarco et al	5	100%	Yes	4	NA	20	80	0	0
1994, Pfeiffer and Cracchiolo	32	100%	Yes	3	10 days	15	29	23	33
1996, Mahan et al	45	NA	Yes	3	NA	24	36	11	29
1997, Turan et al	18	0%	Yes	4	2 weeks	61	22	0	17
1997, Baba et al	34	100%	Yes	3	3 weeks	70	16	8	6
1998, Bailie and Kelitian	36	80%	Yes	3[d]	2 weeks	57	16	11	16
2003, Gondring et al[b]	68	100%	Yes	2?	3 weeks	51[c]	0	0	49
2008, Mullick and Dellon	87	50%	Yes	4[d]	None	82	11	5	2

[Q37] *Abbreviations:* EMG, electromyography; NA; NCV, nerve conduction velocity.
[a] Percentage of patients in the series who did have electrodiagnostic testing. For the Pfeiffer and Cracchiolo series, 81% were positive. For the study by Linscheid et al, 68% were positive. For the study by Baile and Kelikian, 81% were positive. In none of these studies did the nerve conduction velocity/electromyography result correlate with the surgical outcome.
[b] Pneumatic tourniquet was not used.
[c] Outcome: patient-reported improvement. For surgeon-reported pain relief, it was 85% relief of pain and 15% not relieved of pain.
[d] Intertunnel septum was excised.

3.6 mm Hg (range: 0–10 mm Hg) in neutral to a mean of 30.2 mm Hg (range: 3–73 mm Hg) with the ankle pronated and flexed ($P < .001$), whereas, by contrast, the mean pressures in the tarsal tunnel changed from a mean of 3.5 mm Hg (range: 1–6 mm Hg) in neutral to a mean of 15.3 mm Hg (range:1–36 mm Hg). The pressure increase in the medial plantar tunnel with ankle pronation and flexion was significantly greater than in the tarsal tunnel ($P < .001$) and increased to absolute pressure levels able to diminish arterial blood flow within the tibial nerve. Return of these pressures to normal with ankle movement required division of the roof of the medial plantar tunnel. Similar results were found for the lateral plantar tunnel pressure changes with ankle position movement and tunnel release. In some cadavers, excision of the septum between the two tunnels was required to prevent pressures from elevating with ankle pronation and flexion (**Fig. 2**).[23]

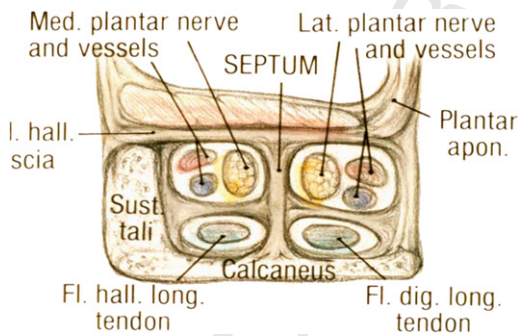

Fig. 1. Cross section through the region immediately distal to the tarsal tunnel demonstrates that there is a medial plantar tunnel and a lateral plantar tunnel, with a fibrous roof. The tunnels are separated by a septum. The abductor hallucis overlies the roof. It is within these tunnels, rather than the tarsal itself, that the pressure critical to symptoms of tibial nerve compression occurs. Apon.■■■; dig.■■■; Fl.■■■; hall.■■■; Lat.■■■; long.■■■; Med ■■■. (*Courtesy of* The Dellon Institutes for Peripheral Nerve Surgery [www.dellon.com], Baltimore, MD; with permission.)

With four medial ankle tunnels, the author designed an operation to decompress the four medial ankle tunnels, as illustrated in **Fig. 3**. A conclusion can be drawn from the meta-analysis given in **Table 1** with regard to the effect of postoperative immobilization after decompression of the tarsal tunnel.[22] The earliest approaches to rehabilitation after tarsal tunnel decompression required ankle immobilization and use of crutches. It is known that during the first weeks after surgery, fibrin deposition is replaced by collagen formation

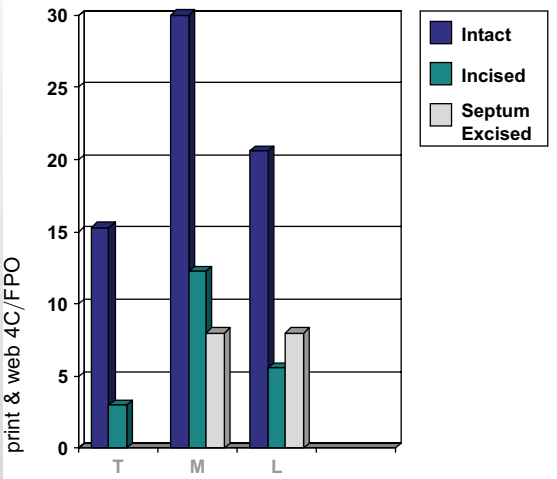

Fig. 2. Changes in pressure (mm Hg on the *Y*-axis) within medial ankle tunnels (T, tarsal tunnel; M, medial plantar tunnel; L, lateral plantar tunnel) with the ankle positioned in pronation and flexion. Note the pressure is highest in the medial plantar tunnel with the tunnels intact. After the roof of the tunnel has been released, the pressure within the tunnel returns to normal in most cadaver specimens; however, for some, the septum between the medial and lateral plantar tunnels also had to be excised. The change in pressure from intact to released was significant at the $P < .001$ level. For the medial plantar tunnel, the further decrease in pressure obtained after excision of the septum was significant at the $P < .02$ level. (*Data from* Barker AR, Rosson GD, Dellon AL. Pressure changes in the medial and lateral plantar, and tarsal tunnels related to ankle position: a cadaver study. Foot Ankle Int 2007:28:250–4.)

and cross-linking, which lead to adherence of the nerve to the surgical site.[24] In contrast, early mobilization of an extremity permits the nerve to glide through the surgical bed, as has been shown for transposition of the ulnar nerve into an intramuscular or submuscular environment in the baboon.[25] Postoperative instructions should include early ambulation, weight bearing, and using a walker to minimize tension on the ankle incision while permitting gliding of the tibial nerve and its branches.[26] Those reported therapeutic level IV studies that immobilized the ankle for 2 to 3 weeks reported the highest percentage of poor plus failed results (14%–49%) regardless of the number of medial ankle tunnels decompressed,[18,27–30] whereas combining a release of four tunnels and permitting immediate mobilization gave the highest percentage of excellent (82%) and the lowest percentage of poor plus failed results (7%):

> *It can be concluded from the evidence that the pressure causing symptoms of tarsal tunnel syndrome is within the medial and lateral plantar tunnels as well as the tarsal tunnel, and that treatment of tarsal tunnel syndrome must include, in addition to opening the tarsal tunnel itself, release of the medial and lateral plantar tunnels and excision of the septum between them to reduce pressure upon the tibial nerve and its branches. Pressure measurements have not yet been obtained for the calcaneal tunnel.*[26]

ELECTRODIAGNOSIS OF TARSAL TUNNEL SYNDROME

In 1965, within 3 years of the first clinical reports of tarsal tunnel syndrome, the *New England Journal of Medicine* published an article on its diagnosis.[31] Forty years ago, the first paper reporting the electrodiagnosis of tarsal tunnel syndrome was published.[32] It was not long before normative data were published for motor[33] and sensory[34] electrophysiology of the medial and lateral plantar nerves, with attempts being made to standardize the recording sites. It became clear that the life events of subjecting the feet to repetitive trauma create a large population of asymptomatic people who have significant electrodiagnostic abnormalities present in these nerves, however. For example, one study demonstrated that 33% of asymptomatic people older than 55 years had absent medial plantar sensory conduction and 50% had electromyographic evidence of denervation in intrinsic muscles.[35] In another study, the extensor digitorum brevis and abductor digiti minimi muscles were examined bilaterally with electromyography in 53 healthy subjects. In 72% of these subjects, fibrillation potentials, positive sharp waves, or fasciculation was seen in at least one muscle examined.[36]

In 2005, the American Association of Neuromuscular and Electrodiagnostic Medicine published a systematic evidence-based review of electrodiagnostic evaluation of patients who had tarsal tunnel syndrome.[37] Of 317 articles published in English from 1965 through 2002, from the National Library of Medicine MEDLINE database, only 4 articles met five or six of the six selection criteria required to meet class III level of evidence. Inclusion criteria for the clinical diagnosis of tarsal tunnel syndrome required typical symptoms by history, with the physical findings required to have "a positive Tinel sign, altered sensation, and weakness of foot muscles." The systematic review concluded that the results of nerve conduction studies were abnormal in some patients who were suspected of having tarsal tunnel syndrome. The sensitivity of needle electromyographic abnormalities could

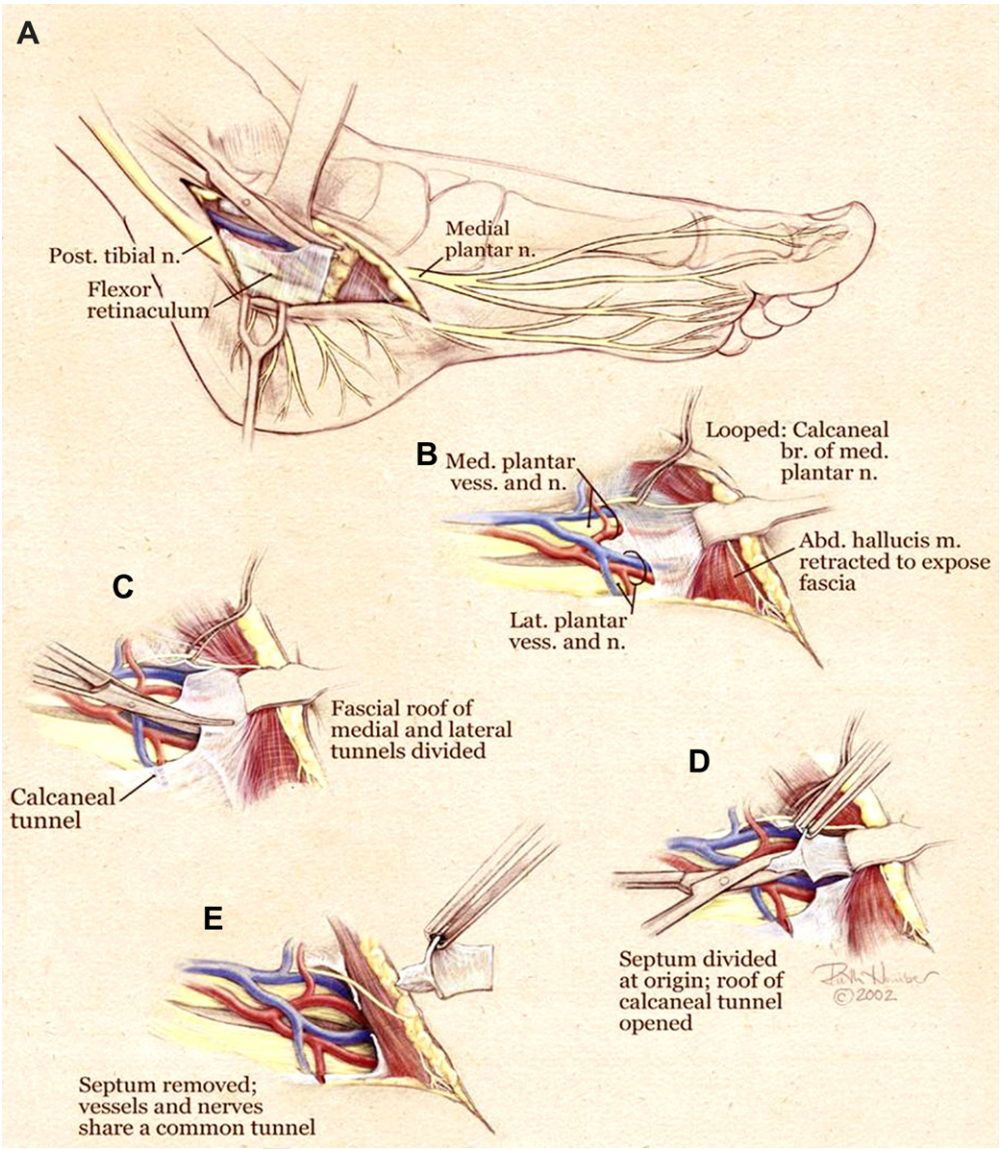

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Fig. 3. An operation designed to decompress the four medial ankle tunnels. (*A*) The tarsal tunnel is opened to identify the presence of any space-occupying lesions and to identify the anatomic variations in the tibial and calcaneal nerves. (*B*) The abductor hallucis muscle is retracted. The branch from the medial plantar nerve to the skin of the most medial heel (usual location for plantar fasciotomy) is protected. (*C*) The roof of the medial plantar tunnel is incised. The roof of the lateral plantar tunnel is incised. (*D*) The septum between the two tunnels is removed. (*E*) The roof of the calcaneal tunnel(s) is incised. (*Courtesy of* The Dellon Institutes for Peripheral Nerve Surgery [www.dellon.com], Baltimore, MD; with permission.)

not be determined. Although sensory nerve conduction studies were more likely to be abnormal than motor ones, the actual sensitivity and specificity could not be determined. It was concluded that nerve conduction studies may be useful for confirming the diagnosis of tarsal tunnel syndrome, with a recommendation for quality of evidence being only of level C, and that well-designed studies were still needed to evaluate more definitely the role of electrodiagnostic testing in patients who have this syndrome:

> *Electrodiagnostic testing cannot easily identify the presence of tarsal tunnel syndrome due to the high percentage of asymptomatic people who have abnormal sensory and motor results.* [Q12]

ELECTRODIAGNOSIS OF TARSAL TUNNEL SYNDROME IN NEUROPATHY

Neuropathy is defined here as a large-fiber, distal, diffuse, sensorimotor, symmetric type. If it is not possible to use electrodiagnostic testing to identify the presence of tarsal tunnel syndrome in otherwise healthy patients, is it possible to diagnose the presence of tarsal tunnel syndrome in the presence of a comorbidity like neuropathy attributable to diabetes? There is no evidence available in the literature to answer this question directly; however, we can infer the answer from evidence available for the most common nerve compression in the upper extremity, the carpal tunnel syndrome. In 2002, a study of critical importance was published by the Neurology Department at the University of Toronto and the Diabetes and Biostatistics groups at the Deaconess Hospital in Boston.[38] Carpal tunnel syndrome was found to have a prevalence of 2% in the nondiabetic population, of 14% in the diabetic population without neuropathy, and of 30% in the diabetic population with neuropathy. Statistical analysis demonstrated that electrodiagnostic parameters are not significant predictors of clinical carpal tunnel syndrome in patients who have diabetes. No electrodiagnostic parameters reliably distinguished diabetic patients who have and do not have carpal tunnel syndrome. That study concluded that given the high prevalence of carpal tunnel syndrome in patients who have diabetic neuropathy and given that electrodiagnostic criteria cannot distinguish the patients who have clinical carpal tunnel syndrome from those who do not have carpal tunnel syndrome and neuropathy, therapeutic decisions for carpal tunnel syndrome should be made independently of electrodiagnostic findings:

> *The evidence has proven that for the carpal tunnel syndrome, electrodiagnostic testing cannot reliably identify the presence of this common upper extremity nerve compression in the patient with an underlying neuropathy, like diabetic polyneuropathy. Extrapolation of this evidence to the lower extremity, where electrodiagnostic evaluation cannot reliably identify the presence of tarsal tunnel syndrome in the patient without neuropathy, suggests that electrodiagnostic studies cannot reliably identify the patient with tarsal tunnel syndrome who also has diabetic polyneuropathy.*[38]

[Q13]

CLINICAL DIAGNOSIS OF CARPAL TUNNEL SYNDROME

It is instructive to begin with the clinical diagnosis of the most common nerve compression in the human body. The clinical diagnosis of carpal tunnel syndrome includes an appropriate history and physical examination. Among surgeons caring for patients who have carpal tunnel syndrome, reliance is placed on provocative signs, such as the Tinel sign (radiation distally along the course of the median nerve when the median nerve is percussed with the examiner's finger)[39] or the Phalen sign (production of symptoms of median nerve compression with wrist flexion). In 2001, a systematic review of the evidence from 1966 through 1999 (42 articles fit the criteria) compared the clinical symptoms and physical findings in patients who had carpal tunnel syndrome with positive electrodiagnostic findings of median nerve compression at the wrist.[40] Only the symptoms of decreased sensation, a drawing of the symptom's location on the hand, or weak thumb abduction correlated with electrodiagnostic testing for carpal tunnel syndrome, whereas a positive Phalen sign or a positive Tinel sign did not correlate with electrodiagnostic findings. These conclusions must be understood in the context of the difficulty with electrodiagnostic testing to identify the presence of carpal tunnel syndrome. A systematic review by the American Association of Electrodiagnostic Medicine, the American Academy of Neurology, and the American Academy of Physical Medicine, published in 2002, documented electrodiagnostic testing to have a false-negative rate of 33%.[41]

A 2002 systematic review from Europe (Poland),[42] where the Tinel sign is referred to as the Hoffmann-Tinel sign because Hoffmann described the identical physical finding in the same year,[43] 1915, as did Tinel,[44] found these provocative tests to be valuable. Using clinical symptoms rather than electrodiagnostic testing as the "gold standard," the Phalen sign had a sensitivity ranging from 42% to 85% and a specificity ranging from 55% to 98%, whereas the Hoffmann-Tinel sign had a sensitivity ranging from 38% to 100% and a specificity ranging from 55% to 100%. In a group of their own patients who had clinical carpal tunnel syndrome, these investigators found that those patients with "false- negative" Phalen and Hoffmann-Tinel signs were those patients with the longest history of symptoms, the more advanced group of patients who had median nerve compression. Confirming this observation is a study from Italy in 2001, which demonstrated in patients who had carpal tunnel syndrome alone and in those who had carpal tunnel syndrome plus neuropathy that the Phalen and Tinel signs were the least sensitive in the patients with the most severe degree of nerve compression.[45] These researchers went on to conclude that this variation in the sensitivity related to clinical and

electrodiagnostic criteria for severity of the nerve compression was one of the reasons for the many contradictions in the literature about these provocative tests. Another instructive review, from 2003, documents that in patients who have clinical carpal tunnel syndrome, electrodiagnostic testing does not predict prognosis. Postoperative electrodiagnostic testing, although usually improved from preoperative testing, does not correlate with the patient's perceived outcome:

> *Clinical diagnosis of carpal tunnel syndrome requires a typical history and a physical examination that demonstrates positive provocative testing, the Tinel or Phalen sign. These signs vary in sensitivity and specificity related to the degree of compression (stage of the disease) of the distal median nerve. Documentation of sensory abnormality also must be documented.*[46]

PATHOPHYSIOLOGY OF THE HOFFMANN-TINEL SIGN

It is suggested that the patient's "positive" response to percussion over a peripheral nerve indicates that axon sprouts are regenerating in this region, whether the nerve is one that has been surgically repaired or one that is "repairing itself" during nerve compression:

> *In…mild nerve compression, the Tinel sign would be negative… [When] the sensory disturbance becomes persistent… The majority of these patients will have a positive Tinel sign… In advanced compression, with…atrophy and loss of two-point discrimination, Tinel sign is often negative because no further regeneration is occurring.—From Dellon AL. Tinel or not Tinel. J Hand Surg [Br] 1984;9:216; with permission.*

With regard to the pathophysiology of the Tinel sign in nerve compression, the previously described rat[13] and monkey[14] models are instructive.[47] Early in nerve compression, when there is decreased oxygen tension within the nerve, most likely there are only paresthesias and no clinical changes are apparent. With beginning demyelination, there are likely to be changes consistent with abnormal cutaneous perception thresholds for vibration and touch.[48–51] With chronicity, and development of axonal loss, there are further elevations in the cutaneous thresholds, but also, for the first time, loss of innervation density, which can be measured by two-point moving and two-point static-touch measurements.[52] The Pressure-Specified Sensory Device (Sensory Management Services, LLC, Baltimore, Maryland) was designed to measure the cutaneous pressure threshold at which distance one from two moving or static prongs contacted the skin.[53–55] This known pathophysiology has measurable sensory changes in the skin target territory of the compressed nerve,[55–57] and this can be used to stage the degree of nerve compression.[58,59] Recently, axonal sprouting at sites of demyelination has been demonstrated[60,61] with this model in the rat.[13,62] Histopathologic examination of human specimens of the chronically compressed superficial sensory radial nerve and the tibial nerve from the tarsal tunnel exhibit these same areas of demyelination and sprouting.[63,64] It may be inferred that the Tinel sign represents signaling from these mechanically sensitive sprouts at the sites of chronic nerve compression and that, with the more advanced degrees of nerve compression, sprouting may have stopped, giving the apparent "false-negative" response in the patient who has advanced carpal tunnel syndrome.

CLINICAL DIAGNOSIS OF TARSAL TUNNEL SYNDROME

Research is now appearing with regard to provocative testing in patients who have clinical tarsal tunnel syndrome similar to that described previously for carpal tunnel syndrome. In 2001, in an attempt to add "objectivity and consistency" to the diagnosis of tarsal tunnel syndrome, an examination technique similar to the Phalen test was introduced.[65] In this test, the ankle is passively maximally everted and dorsiflexed, whereas all the metatarsophalangeal joints are maximally dorsiflexed and held in this position for 5 to 10 seconds. The test was done on 50 normal volunteers (100 feet) and on 37 patients with symptoms typical for tarsal tunnel syndrome, in whom 7 had bilateral symptoms (44 feet). These 44 feet were treated by surgery for tarsal tunnel syndrome between 1987 and 1997. The dorsiflexion-eversion test was done before and after surgery. The mean postoperative follow-up was 3.8 years. From the author's data, the sensitivity of this test can be calculated to be 97% (43 of the 44 clinically positive patients for tarsal tunnel syndrome had worsening of their symptoms) and the specificity of this test can be calculated to be 100% (none of the 100 feet in the control population responded with symptoms during this test).

A study of the presence of a Tinel sign over the tibial nerve in the tarsal tunnel was reported in 2003.[30] All 68 patients in that study had a positive Tinel sign as a requirement for inclusion in a cohort

to have tibial nerve decompression for tarsal tunnel syndrome. By definition then, the sensitivity of the Tinel sign was 100%, but the specificity cannot be calculated for that study. The positive predictive value of the Tinel sign can be calculated from that study as 85% for complete relief of symptoms at 3 months after surgery. Sensitivity and specificity for the Tinel sign in patients who had tarsal tunnel syndrome are also available from the 2001 study describing the dorsiflexion-eversion test.[65] Sensitivity of the Tinel sign was 92%, and specificity was 100%. It should be recalled that the American Association of Neuromuscular and Electrodiagnostic Medicine, which published in 2005 a systematic evidence-based review of electrodiagnostic evaluation of patients who had tarsal tunnel syndrome,[37] required a positive Tinel sign to be present for a patient with a typical history of tarsal tunnel to be included in its review:

> *Clinical diagnosis of tarsal tunnel syndrome requires a typical history and a physical examination that demonstrate positive provocative testing, the Tinel sign or dorsiflexion-eversion test. These may vary in sensitivity and specificity related to the degree of compression (stage of the disease) of the distal tibial nerve. Documentation of sensory abnormality also must be provided.*[37]

NEUROPATHY AND NERVE COMPRESSION

Neuropathy, in the current context, has been well characterized[66,67] and is here defined as a large-fiber, distal, diffuse, symmetric sensorimotor disease and is most often identified with diabetes mellitus. For the purposes of this discussion, this can be called diabetic polyneuropathy (DPN). In some patients, small nerve fibers can be involved, making this a mixed form of neuropathy; certainly, if there is a superimposed nerve compression, the small myelinated and unmyelinated fibers can become involved by the compression. DPN is manifested in the upper and lower extremities in the classic ''stocking and glove'' pattern. It can be associated with pain and can have involvement of small myelinated and unmyelinated fibers demonstrated on skin biopsy, but there must be demonstrated large-fiber abnormalities as shown by quantitative measurement of the cutaneous pressure or vibratory threshold, or by electrodiagnostic testing.[68,69] The natural history of DPN is well documented and well known, is unchanged, and remains ''progressive and irreversible,''[70] with a predictable number of patients with admission to the hospital for foot infection, ulceration, amputation, loss of balance, and falls associated with fractures of the hip and wrist and occult fractures of the insensate foot.[71–74] There is currently no known preventative treatment for DPN. Even with the frequent monitoring of blood glucose and tight control, neuropathy is still prevalent in approximately 18% of the population.[75] In the absence of tight control, and depending on the methodology used to identify the presence of DPN, approximately 50% or more of diabetics develop DPN within 15 to 20 years of the onset of their disease. For those with painful DPN, there is the progression of neuropathic pain medication and then opiates.[76] Diabetes has reached epidemic proportions; therefore, so too has DPN.[77] There may be 10 million people within the United States today with this problem. Eugene Barrett, MD, past president of the American Diabetes Association, said in his Presidential Address in 2004 that the cost of caring for diabetes mellitus alone will bankrupt the Medicare Trust Fund.[78]

There are, of course, other similar neuropathies that occur in patients who do not have diabetes. The American Peripheral Neuropathy Association estimates there are as many patients who have neuropathy who do not have diabetes as there are with diabetes. Chemotherapy-induced neuropathy, attributable to agents containing platinum or Taxol, and now thalidomide (for multiple myeloma) is increasing. The incidence of disabling neuropathy (grades 3 and 4) occurs in 8% of those patients who have breast cancer and are receiving weekly paclitaxel, with grade 2 adding another 19%[79] The epidemic in obesity has created a population of patients with glucose intolerance.[80] It was first reported in 1999 that hyperinsulinemia, present in those with metabolic syndrome, is related to neuropathy.[81] It is now clear that approximately 56% of patients who have idiopathic neuropathy, if tested for impaired glucose tolerance, are found to fit into this category,[82] putting them at risk for the complications associated with DPN.

The metabolic mechanisms of some forms of neuropathy can predispose the peripheral nerve to chronic compression. For example, in diabetes, the polyol pathway converts, by means of aldose reductase, glucose into sorbitol. Sorbitol is hydrophilic and causes water to come into the nerve, creating endoneurial and subperineurial edema.[83] Furthermore, in diabetes, the slow anterograde component of axoplasmic transport is reduced.[84] With platin and Taxol neuropathy, it is known that these agents bind to tubulin within the peripheral nerve, causing the slow anterograde component of axoplasmic transport to be reduced.[85] This subject has been reviewed in depth.[86] It was

hypothesized in 1973 that a proximal constraint on axoplasmic flow or an underlying metabolic neuropathy could predispose the peripheral nerve to more distal entrapments.[87] This was demonstrated in a rat model[88] and then in a streptozotocin-induced diabetic rat model.[9]

Results suggest that approximately one third of patients who have neuropathy have chronic nerve compressions. In a population of Canadians with diabetes, 14% of those without neuropathy and 30% of those with neuropathy had carpal tunnel syndrome.[38] In another study evaluating upper and lower extremity sites, 33% of the patients were found to have a chronic nerve compression.[89] In the upper extremity, these were found to be carpal tunnel syndrome, cubital tunnel syndrome, and radial nerve entrapment, and in the lower extremity, these were found to be entrapment of the common peroneal nerve and tarsal tunnel syndrome.

If you combine the skin territories for three separate nerve entrapments in the upper extremity, the median, ulnar, and radial nerves, you would get the pattern of a glove. If you were to combine the skin territories for the peroneal and tibial nerves in the lower extremity, you would get the pattern of a stocking.

Is it possible that some of the symptoms in the patients who have neuropathy are attributable to the presence of nerve compressions? If the answer is "yes," relief of these symptoms would be possible with surgery by decompression of those nerves. This was first expressed in this context in 1988,[90] when it was stated there may be a new optimism for those with neuropathy if their compressed peripheral nerves could be decompressed:

> *Neuropathy related to diabetes, impaired glucose tolerance, and chemotherapy predisposes the peripheral nerve to chronic nerve compression. Chronic nerve compression is prevalent in patients with diabetes. Multiple nerve compressions in the same patient would give the appearance of a stocking or glove pattern of sensory impairment. It is therefore possible that decompression of a peripheral nerve in a patient who has both neuropathy and chronic nerve compression can relieve symptoms related to that particular nerve.[90]*

EVIDENCE FAVORS DECOMPRESSION IN EXPERIMENTAL NEUROPATHY

In the streptozotocin-induced diabetes rat model, a progressive neuropathic walking track pattern develops consistently.[91] This pattern improves toward normal if the blood sugar returns from 400 to 90 dcl/cm^3. If the blood sugar is maintained at the 400 dc/cm^3 level, two groups of rats are compared (one group with a normal tarsal tunnel anatomy and one group in which the compressive sites at the medial ankle have been surgically removed), and the animals are followed for 1 year (half of their life expectancy), the neuropathic walking track pattern then develops as expected in the group with the normal tarsal tunnel. The animals without a site for chronic nerve compression walk with a pattern exactly the same as weight-controlled nondiabetic rats with a tarsal tunnel, however.[92] This study was repeated by a separate group of surgeons in Turkey, who found the same results. In addition, they identified that adding internal neurolysis to the nerve decompression gave additional significant functional improvement to decompression of the tibial nerve alone.[93] A similar study was repeated by a group of surgeons from the Cleveland Clinic.[94] This study used a Zucker rat model and added pinprick, muscle weight, and somatosensory-evoked potentials to the evaluation procedures in addition to the walking track analysis. They found the same result with the addition that combining decompression of the peroneal nerve with the tarsal release had additional significant functional improvement.

A similar study was done in a group of rats that developed a neuropathic walking track pattern after receiving cisplatin chemotherapy.[10] In those animals that did not spontaneously revert to a normal walking track pattern after cessation of chemotherapy, surgical decompression of the tarsal tunnel permitted functional improvement of a normal walking track pattern:

> *Experimental evidence demonstrates that, in the absence of a site of compression, neuropathy, as documented by a walking track model in the rat, does not develop despite severe hyperglycemia.*
>
> *Experimental evidence demonstrates that decompression of the tibial nerve in the hyperglycemic diabetic rat model improves function as documented by walking track analysis. Function is improved in the rat model of cisplatin neuropathy by decompression of the tibial nerve.*
>
> *Experimental evidence demonstrates that decompression of the peroneal nerve plus the tibial nerve adds to the functional improvement recovered in the hyperglycemic rat model.[10]*

EVIDENCE FAVORS DECOMPRESSION IN CLINICAL NEUROPATHY IN PATIENTS WHO HAVE CHRONIC NERVE ENTRAPMENT

For this section, a review of the literature and papers presented at national meetings was evaluated. Twenty-two studies were identified and are discussed. It must be emphasized that although in the experimental models of neuropathy, anatomic sites of nerve compression were decompressed in every animal without identifying a localizing sign of compression, in this clinical section, only those patients who had neuropathy that also had one or more coexisting chronic nerve compressions were included in the surgical cohorts. This review does not include any peer-reviewed article whose inclusion criterion for surgery is neuropathy alone. Only patients identified as having a nerve compression by the presence of a positive Tinel sign and wgi also had neuropathy were included in the studies.

Retrospective Level IV Therapeutic Studies

The results of the first retrospective level IV therapeutic study were published in 1992. (**Table 2**). [95] There were 60 diabetic patients: 28 type I and 32 type II. Multiple peripheral nerves were decompressed in 51 upper and 31 lower extremities, for a total of 154 nerves. To be included in this study, each patient had to be under good glycemic control, to have failed a medical regimen for symptomatic relief, and to have a positive Tinel sign over the site of anatomic narrowing (nerve compression site). For this study, 94% of the patients had electrodiagnostic testing. These demonstrated that 8% were ''normal,'' 11% had a single nerve entrapment, 43% had diffuse neuropathy with superimposed nerve entrapment, and 38% had diffuse neuropathy without nerve entrapment. The mean follow-up was 30 months (range: 6–83 months). Outcome measures used were as follows:

1. Electrodiagnostic (100% of those originally diagnosed as having localized compression were improved, 80% of those originally diagnosed with diffuse neuropathy plus nerve compression were improved, and 55% of those originally diagnosed as having diffuse neuropathy were improved)
2. Subjective (88% improved, 10% not improved, 2% worse)
3. Development of ulceration or amputation (none developed)
4. Observation of the unoperated contralateral limb (50% of these demonstrated the expected progression of their neuropathy. This last observation was the first evidence that the natural history of diabetic neuropathy could be altered from progressive and irreversible.

Three more retrospective level IV therapeutic studies were published (see **Table 2**).[96–98] Each included patients who were more advanced in their neuropathy than the 1992 study, in that a total of 40 of the 101 patients included in those two studies were patients who had a history of an ulcer or an amputation. These three studies did not require a positive Tinel sign for inclusion. They reported that an average of 89% of the patients improved in their preoperative pain level and that an average of 61% of the patients had improved sensation. Importantly, of the expected more than 50% of the patients who would get recurrent ulceration in this group,[72,73] recurrent ulceration occurred in just 1 patient (2.5%) This was the next evidence that the natural history of diabetic neuropathy, and its complications in terms of wound healing, could be changed if sensibility could be restored to the feet.

Prospective level IV Therapeutic Studies

There have now been nine studies reported in which all surgeons were trained in the Dellon surgical technique, as described previously, to

Table 2
Distal tibial nerve decompression in diabetics with tarsal tunnel syndrome: retrospective therapeutic level IV studies

		Before Surgery		Results: Improved		
Study	No. Nerves	Ulcers	Amputation	Pain	Touch	New or Recurrent Ulceration or Amputation
Dellon, 1992	31	0	0	85%	72%	0%
Wieman, 1995	33	13	0	92%	72%	7%
Chaffe, 2000	58	11	6	86%	50%	0%
Tambwekr, 2001	10	6	4	na	80%	0%

[Q38] *Abbreviation:* na, ■■■.

decompress the four medial ankle tunnels.[99–107] In each of these studies, listed in **Table 3**, the inclusion criteria were the same: patients who had controlled diabetes and did not respond to medical management of their symptoms, who had a positive Tinel sign over the tarsal tunnel, and absence of previous ulceration or amputation. Outcomes were the same in each study: visual analog scale for pain evaluation, measurement of sensibility with the Pressure-Specified Sensory Device, and recording of new ulceration or amputation. These studies were started before the patient enrolled. Historical data were considered sufficient for a comparison group (ie, neuropathy is progressive and irreversible, 2% per year of diabetics develop an ulcer, 15% of diabetics with loss of protective sensation develop an ulceration, 10% of diabetics have an amputation). From **Table 3**, it is clear that in 350 patients who had DPN and a positive Tinel sign over the tibial nerve in the tarsal tunnel, decompression of the four medial ankle tunnels resulted in relief of pain for 80% and improvement in sensation also for 80% of the patients. These patients were all at approximately the same stage of their neuropathy in that none had a previous ulceration or amputation, and none of these patients developed an ulcer or had an amputation after the operation for the period of follow-up, which averaged 12 months per study (range: 3–23 months per study). There were only two types of postoperative complications: small wound healing problems, with none requiring hospital admission (12%,[100] 27%,[101] 10%,[103] 10%[105]), and an occasional patient whose foot had not been painful now becoming painful during neural regeneration and requiring pain medication. The other studies reported no postoperative complications.[99,106,107] [Q17]

Using the same inclusion criteria and methodology as those studies published previously, there have now been six similar studies presented at national meetings but not yet appearing in peer-reviewed journals. These are given in **Table 4**. In these eight presentations are included 425 patients. The results are the same as given in **Table 3** for the published peer-reviewed studies: 80% improvement in pain, 80% improvement in sensation, and no new ulcerations or amputations.

Finally, a pilot study of just six patients who had nine distal tibial decompressions was evaluated with the Short-Form Health Survey (SF-36).[108] That study design suffers from not having tested the patients before surgery, but the researchers compared their few surveys with published data and found that, with the exception of the role-physical and role-emotional categories, their postoperative patients were not different from diabetics who did not have neuropathy, patients who had back pain, and age-matched normal controls. Future prospective studies should include a quality-of-life measure in the outcome assessment.

Retrospective Level III Prognostic Studies

Ulceration and amputation

There are two level III studies that are listed in **Table 5**.

One of the most crucial questions to be asked is whether decompression of peripheral nerves in a patient who has DPN and chronic nerve compressions can change the natural history of DPN in terms of its two most dreaded and costly complications: ulceration and amputation. A criticism of level IV studies that have demonstrated reduction of ulceration and amputation might be that

Table 3
Distal tibial nerve decompression in diabetics with tarsal tunnel syndrome: prospective therapeutic level IV studies

		Before Surgery		Results: Improved		New or Recurrent
Study	No. nerves	Ulcers	Amputation	Pain	Touch	Ulceration or Amputation
Aszmann, 2000	16	0	0	na	69%	0%
Wood, 2003	33	0	0	90%	67%	0%
Biddinger, 2004	22	0	0	86%	80%	0%
Valdivia, 2005	60	0	0	87%	85%	0%
Rader, 2005	49	0	0	90%	75%	0%
Yong, 2005	90	0	0	94%	90%	0%
Siemionow, 2006	36	0	0	90%	90%	0%
Karagoz, 2008	24	0	0	75%	89%	0%
Massa, 2008	20	0	0	80%	86%	0%

Table 4
Distal tibial nerve decompression in diabetics with tarsal tunnel syndrome: prospective therapeutic level IV studies: presentations at national meetings

Study	No. Nerves	Before Surgery: Ulcers	Before Surgery: Amputation	Results: Improved: Pain	Results: Improved: Touch	New or Recurrent Ulceration or Amputation
DiNucci, 2005[a]	72	0	0	80%	80%	0%
Steck, 2005[b]	25	0	0	84%	72%	0%
Maloney, 2005[c]	95	0	0	86%	83%	0%
Shaffiroff, 2006[d]	300	0	0	85%	80%	0%
Bae, 2007[e]	33	0	0	75%	72%	0%

[a] DiNucci K. Results of decompression of multiple lower extremity peripheral nerves in diabetic with symptomatic neuropathy. Presented at the American College of Foot and Ankle Surgery meeting, New Orleans, March 2005.
[b] Steck J. Results of decompression of lower extremity peripheral nerve for treatment of symptomatic neuropathy. Presented at the American Society of Peripheral Nerve meeting, Puerto Rico, January 2005.
[c] Maloney CT, Valdivia JV, Weinand M. Nerve decompression results in a consecutive series of 165 patients with neuropathy. Presented at the Neurological Surgical Society meeting, Spine and Peripheral Nerve section, Phoenix, 2005.
[d] Shafiroff B. Decompression of lower extremity nerves in neuropathy. Presented at the Lower Extremity Peripheral Nerve Surgery meeting, Sante Fe, October 2006.
[e] Bae S, Biddinger K, Shon L. Independent retrospective review of surgical nerve decompression for diabetic neuropathy. Presented at the American Academy of Orthopedic Surgery Foot and Ankle Society meeting, San Diego, February 2007.

these patients were under their best possible glycemic control and that this euglycemia, rather than the decompression, was the source of eduction in neuropathic complications. To evaluate this, in 2004, a group of 50 patients who had DPN and had received only unilateral decompression surgery were interviewed to determine the occurrence of new ulcers and new amputations in the limb that was decompressed and in the nonoperated contralateral limb, with that contralateral limb serving as the case-control ''limb'' of the study and with blood sugar the same in each limb.[109] Inclusion criteria for these patients as surgical candidates were the same as for the studies discussed previously, with the additional inclusion criteria that they had not had bilateral decompression surgery. The mean follow-up was 4.5 years (range: 2–7 years). The ''experimental'' side, the decompressed side, had no ulcers and no amputations. In contrast, the contralateral control side developed 12 ulcerations and three amputations. This difference was statistically significant at the $P < .001$ level. This study suggests that decompression of the distal tibial nerve can alter the natural history of DPN. This study reported in human patients what the three basic science studies in rats had demonstrated.[92–94]

Previous carpal tunnel surgery

The second study evaluated whether clinical success from previous carpal tunnel decompression, an upper extremity peripheral nerve compression, would serve as a predictor of success for decompression of the distal tibial nerve.[110] From a cohort of 300 patients who had the lower extremity decompression for neuropathy, 35 were identified for whom there were data on the outcome of their carpal tunnel decompression.

Of the 35 patients, 34 had a successful outcome after carpal tunnel decompression and 1 did not.

Table 5
Distal tibial nerve decompression in diabetics with tarsal tunnel syndrome: prognostic level III studies

Study	No. nerves	Before Surgery: Ulcers	Before Surgery: Amputation	Results: Improved: Pain	Results: Improved: Touch	New or Recurrent Ulceration or Amputation
Lee, 2004	46	0	0	92%	92%	na
Aszmann, 2004	50	0	0	na	na	0%
Maloney, 2007	38	0	0	88%		0%

Abbreviation: na, ■■■.

Of the 34 patients who had successful carpal tunnel decompression, 30 had a successful outcome from tibia nerve decompression. This study demonstrated an 88% positive predictive value of success from decompression of the four medial ankle tunnels if previous carpal decompression was successful.

Prospective Level II Prognostic Study

Positive Tinel sign

Since 1992, the presence of a positive Tinel sign has been the criterion for consideration of a patient who has neuropathy as a surgical candidate for lower extremity peripheral nerve decompression.[95] Of course, there were patients who did have surgery who did not have a positive Tinel sign based on other considerations, such as severe and debilitating pain and intolerance to medication. It was therefore appropriate to evaluate prospectively a group of patients who had neuropathy and a positive or negative Tinel sign and to relate that to the outcome from the distal tibial nerve decompressions. This study was reported in 2004.[111] At 1 year after surgery, patients were dichotomized into either good/excellent or poor/fair outcomes to be compared statistically with those who had a positive or a negative Tinel sign before surgery. For the 46 patients who had DPN, there was an 88% positive predictive value for a good/excellent outcome. For the 40 patients who had idiopathic neuropathy, there was a 93% positive predictive value for a good/excellent outcome. This study documents the value of a positive Tinel sign at the site of known anatomic narrowing for a given peripheral nerve in predicting a successful outcome for decompression surgery, and therefore in identifying patients who would most benefit from this surgery.

What might be expected for those patients who had a negative Tinel sign? Should they be denied surgery? In 1984, a discussion of the theoretic implications of the Tinel sign in nerve compression was written[112] and establishes the background for interpreting this sign, even as discussed previously. At a known anatomic site of narrowing, a site for nerve compression, a negative Tinel sign does not mean that there is no nerve compression but rather that the degree is advanced with little if any axonal sprouting currently occurring. It may still be possible for decompression at this site to yield a good result, but the success rate cannot be that high. In the study just described,[111] for those patients who had DPN and a negative Tinel sign, 33% still had a good/excellent outcome. For those patients who had idiopathic neuropathy, 28% still had a good/excellent outcome.

Prospective Level II Prognostic Study

Balance

Clinically, it was apparent that as the foot became more insensitive, the patients experienced balance problems manifested by falls. For example, 41% of diabetics with impaired sensation, as determined by means of the Semmes-Weinstein monofilaments, fell once per year, with a mean of 1.25 falls per year.[113] In a recent report, 35% of 150 women with type II diabetes and impaired vibratory perception each had one episode of a fall associated with a fracture.[114] Almost identical findings have been reported this year by another group of investigators.[115] In a level I diagnostic study, evaluation of sensibility with the Pressure-Specified Sensory Device has been demonstrated to be more sensitive than evaluation of sensibility using the Semmes-Weinstein monofilaments or vibration threshold using the Vibrometer (100% versus 63% versus 30% respectively) [Q18] and more specific (100% versus 70% versus 80%, respectively).[116] In 2004, a retrospective study of patients who had neuropathy correlated increasing loss of sensibility, as measured with the Pressure-Specified Sensory Device, with increasing loss of balance, as measured by sway using the MatScan Measurement System (Tekscan, Inc., Boston, Massachusetts).[117] Then, in a prospective study in 2006,[74,118] patients who had neuropathy had their balance measured before surgical decompression of the four medial ankle tunnels, as described previously. Neuropathy was the result of diabetes in 72% of patients, the result of a combination of diabetes and hypothyroidism in 7%, the result of chemotherapy in 7%, and idiopathic in 14%. The mean age of the patients was 67 years. In those patients who had bilateral staged decompression, there was an overall significant improvement in sensibility compared with their preoperative sensibility ($P < .004$) and in their balance ($P < .02$). Outcome in terms of reduction of fracture risk can be obtained from the multicenterNeuropathyRegistry.com prospective study, in which 1182 patients at 1 year after decompression had no recorded fractures.[119] [Q19]

Prospective Level II Therapeutic Study

Multicenter clinical outcomes

A prospective comparative study was initiated as a multicenter study to make available to the public on-line clinical results that could be compared with historic controls. Clicking on ''Statistics'' on

theNeuropathologyRegistry.com Web site brings up a menu of outcomes that include, pain, recovery of sensation, new amputations, new ulcerations in those patients with or without a previous history of ulceration, and hospitalizations for foot infections. At present, 39 surgeons have contributed to this database. Each of the surgeons has been trained in the same surgical technique discussed previously and uses the same inclusion criteria as the previously published studies, with the exception that patients can have a previous ulceration or toe amputation and still be a candidate for surgery if there is a positive Tinel sign. As of April 25, 2008, the site had recorded 1530 patients who had neuropathy and had undergone 1181 operations (351 had the contralateral side decompressed). Of these 1530 patients, 619 were diabetics. The results are displayed by means of Kaplan-Meier proportional hazards. **Fig. 4** is an example of the comparative analysis for patients who have DPN and no previous history of ulceration, with the expected 15% ulceration compared with the actual level of 0.3%. **Fig. 5** is an example of the same analysis for DPN with a previous history of ulceration, with the expected 50% recurrent ulceration rate compared with the actual level of 3.8%. A final example is particularly instructive in view of the recent article by Lavery and colleagues regarding hospitalization for foot infections in a population of 1666 patients who had DPN and were receiving "optimal" foot care for 2 years.[112] There were 9.1% infections, and 3.7% of affected patients were admitted to the hospital. In comparison, theNeuropathyRegistry.com has 869 patients followed for 1.5 years with 0.8% admissions for foot infections:

[Q20]

There is evidence at every level, except Level 1, that, in the patient with neuropathy, decompression of a compressed lower extremity peripheral nerve, has an 80% chance to greatly relieve pain, an 80% chance to improve sensation, and thereby greatly reduce expected incidence of ulceration and amputation in this patient population.

The site of compression in these studies was determined by the presence of a positive Tinel sign at a known site of anatomic narrowing.

These studies demonstrate that the natural history of diabetic polyneuropathy can be changed, and thereby lies the potential to improve health care outcomes and health care costs.

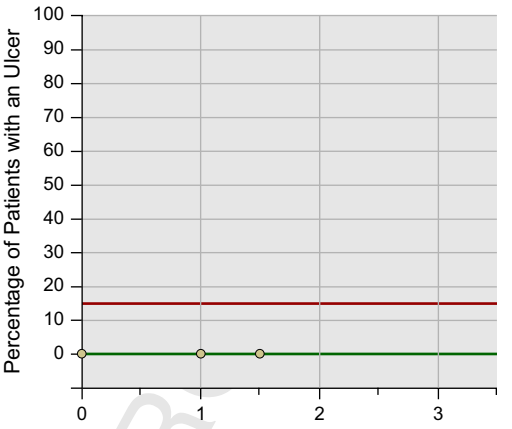

Fig. 4. Graphic analysis of patients who have DPN and no previous history of ulceration. Each has had decompression of the four medial ankle tunnels. Instead of the expected 15% incidence of ulceration historically found in this population, successful restoration of sensation has reduced the incidence of ulceration to 0.3%. (*Courtesy of* The International Neuropathy Decompression Registry [http://neuropathyregistry.com], Baltimore, MD; with permission.)

There is evidence that this approach can be successful in patients with neuropathy not due to diabetes, such as chemotherapy-induced neuropathy and idiopathic neuropathy with impaired glucose tolerance.

[Q21]

DISCUSSION

The basic science and clinical evidence reported here are of good quality (level A),[1] with observations being confirmed by multiple investigators from around the world and from many different surgical subspecialties. There would seem to be little room for disagreement about the essential components of these concepts:

1. Neuropathy predisposes an individual to chronic nerve compression.
2. Chronic nerve compression can be identified clinically by history, physical examination, and the presence of a positive Tinel sign at a known site of anatomic narrowing.
3. Sensibility can be measured in patients who have neuropathy and nerve compression.
4. Chronic nerve compression can be treated by decompression of the involved nerve(s) with appropriate surgical technique and skill.
5. Outcomes of nerve decompression can be evaluated for pain reduction, improvement in

[1] Level A: good scientific evidence suggests that the benefits of the clinical service substantially outweigh the potential risks. Clinicians should discuss the service with eligible patients (http://en.wikipedia.org/wiki/Evidence-based_medicine).

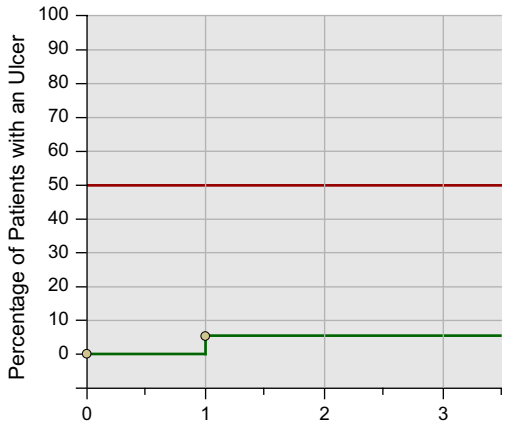

Fig. 5. Graphic analysis of patients who have DPN and a previous history of ulceration. Each has had decompression of the four medial ankle tunnels. Instead of the expected 50% incidence of recurrent ulceration historically found in this population, successful restoration of sensation has reduced the incidence of ulceration to 3.8%. (*Courtesy of* The International Neuropathy Decompression Registry [http://neuropathyregistry.com], Baltimore, MD; with permission.)

sensibility, development of ulceration, development of amputation, occurrence of falls with and without fractures, and admission to the hospital for infection. The only complications reported in the studies in **Table 3** are minor wound healing in approximately 8% to 12% of the patients in the medial ankle incision group.

It must be emphasized that in all the studies cited here with respect to this approach, only patients with chronic nerve compression and the comorbidity of diabetes, chemotherapy,[120,121] or idiopathic neuropathy[102,111] (many of whom have impaired glucose tolerance[82]) have had nerve decompression. No suggestion has been made that surgical decompression should be attempted in all patients who have neuropathy.

Although this article has addressed the decompression of the distal tibial nerve, it should be emphasized that the clinical reports in **Table 2** include decompression of the common peroneal nerve[122,123] at the knee and the deep peroneal nerve at the dorsum of the foot,[124] because the peroneal and the tibial nerve skin territories are required to comprise a stocking pattern. A positive Tinel sign was present at both sites of the peroneal nerve compression. Interestingly, a study of electrodiagnostic screening has found that peroneal conduction across the fibular neck correlated with identification of diabetics with symptomatic neuropathy, and evaluation of the common peroneal nerve was suggested to be part of the screening examination for the primary care physician.[125] It is beyond the scope of this present article to discuss further peroneal nerve entrapment sites, which include, less often, the superficial peroneal.[126] Importantly, the recommendation by Vinik[127] that individual sites of chronic nerve compression be "un-entrapped" further supports this approach to patients who have neuropathy in whom an entrapment site can be demonstrated.

In 2006, the American Academy of Neurology reviewed some of the published clinical evidence presented in the present article and discussed, for example, the publications listed in **Tables 3** and **4**. This review is entitled "Practice Advisory: utility of surgical decompression for treatment of diabetic neuropathy: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology".[128] As indicated previously, and as explicitly stated in this article, the emphasis in this review is not the "treatment of diabetic neuropathy" by "surgical decompression." Because the stated purpose of the Practice Advisory was not the intent of any of the papers reviewed, it is not surprising that the American Academy of Neurology concluded that:

> *Systematic review of the literature revealed only Class IV studies concerning the utility of this therapeutic approach. Given the current evidence available, this treatment alternative should be considered unproven (Level U). Prospective randomized controlled trials with standard definitions and outcome measures are necessary to determine the value of this therapeutic intervention.*[128]

In the first reply to this article by the American Academy of Neurology, Peter J. Dyck, MD, Chief of the Peripheral Nerve Section of the Department of Neurology of the Mayo Clinic, whose work related to diabetic neuropathy over the past 40 years has been referred to already,[66,67] wrote, "It should be emphasized that it is decompression of leg nerves at anatomic sites not known to be entrapped that is being discussed [by the Practice Advisory] (which may not have been sufficiently emphasized in the Advisory)."[129] Dyck is stating that the review does not apply to compressed nerves in the patient who has neuropathy but to neuropathy in general, because he clearly perceives this difference in the published papers' intent compared with the title of the American Academy of Neurology's review.

Is a randomized clinical trial necessary to determine if nerve decompression is efficacious in a patient with one or more nerve entrapments [Q22]

and a comorbidity like neuropathy? The Practice Advisory[128] and commentary[37,130,131] have made this recommendation. A randomized clinical trial is a method of comparing two therapeutic modalities. Yet, there is no question, even among those who have participated in the commentary,[130] such as A.I. Vinik, that nerve compression in the diabetic should be decompressed.[89,127] If there were another therapy with which to compare the nerve decompression of a compressed nerve in the patient who has neuropathy, it would be appropriate to do so in a randomized control trial, but as reviewed previously, and as noted by others,[132] DPN is progressive and irreversible and without a known treatment other than attempted euglycemia and neuropathic pain medication. The writers of the Practice Advisory[128] and the correspondence[129] suggest that a surgery placebo group would be appropriate, in that surgery itself can have a placebo effect. Surgery may well have a placebo effect, but even if it had a placebo effect of 30%, the observed magnitude of the improvement in pain and sensory recovery, a magnitude of 80%, would easily be shown to be a significant improvement with a relatively small number of patients, approximately 30, in each group of such a study. There remains the question of whether an ethics committee and the institutional review board would approve of a sham surgical control group in patients who have diabetes, given their risk for cardiovascular events and wound healing problems. Randomized trials are useful to identify the effect of a procedure on groups that may be omitted from observational studies, thereby creating a bias. Nevertheless, one might ask, ''Who has been excluded'' from the observational studies reported previously. Those studies contain whites, Hispanics, African Americans, and Asians; men and women; type I and type II diabetics; and age ranges from 25 to 80 years, with surgeons from the United States, Turkey, and China in addition to surgeons who are in private practice and academic practice, hand surgeons, neurosurgeons, orthopedic surgeons, podiatric foot and ankle surgeons, and plastic surgeons. Only patients with impaired circulation or foot edema were excluded, and these exclusions are medically indicated. Perhaps a randomized controlled clinical trial in which appropriate patients are allocated to surgery versus best medical care would address the Practice Advisory panel's concerns. This would avoid the ethical and medical complications associated with sham surgery. Such a study would at best be single-blinded (to an independent outcome measurer) but could eliminate much of the bias inherent to nonrandomized studies:

Physicians must make clinical decisions based upon the best available evidence. The existing evidence is of good quality and demonstrates that decompression of peripheral nerves in the lower extremity of a patient with neuropathy, who also has evidence of one or more nerve compressions, can change the natural history of diabetic neuropathy by restoring sensibility, relieving pain, restoring balance, preventing ulceration, minimizing hospitalization for foot infections, and preventing amputation.

[Q23]

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